Supreme Court, U.S.
FILED
MAR 4 - 2015
OFFICE OF THE CLERK

Nos. 14-46, 14-47, and 14-

IN THE

Supreme Court of the United States

STATE OF MICHIGAN, ET AL.,
PETITIONERS,

v.

ENVIRONMENTAL PROTECTION AGENCY, ET AL.,
RESPONDENTS.

On Writs of Certiorari to the United States Court of Appeals for the District of Columbia Circuit

BRIEF OF AMICI CURIAE HEALTH SCIENTISTS, DR. LYNN GOLDMAN ET AL, IN SUPPORT OF RESPONDENTS

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March 4, 2015

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INTERESTS OF THE AMICI[1]

Each of the amici is an expert in environmental public health. Dr. Lynn Goldman is the Dean of the Milken Institute School of Public Health and Professor of Environmental and Occupational Health at George Washington University. A pediatrician and an environmental epidemiologist, she has an MD, a Masters of Science in Health and Medical Sciences, and a Masters of Public Health. She has served as the Assistant Administrator for Toxic Substances at the Environmental Protection Agency, a professor of Environmental Health Sciences at the Bloomberg School of Public Health, and a Public Health Medical Administrator with the California Department of Public Health. The other 15 health science experts who are amici are listed in the Addendum to this brief, along with their specific areas of expertise and their current affiliations.

INTRODUCTION AND SUMMARY OF ARGUMENT

The issue before this Court is whether the respondent Environmental Protection Agency (EPA) properly decided to regulate emissions of hazardous air pollutants from power plants without considering the costs imposed by such regulation at the initial listing stage. This brief

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[1] This brief is filed based on the blanket consents of all parties on file with the Court. No counsel for a party authored this brief in whole or in part, and no one other than the George Washington University, the amici, or their counsel contributed money that was intended to fund the preparation or submission of this brief.

will focus on the acute and chronic dangers to public health caused by the exposure of mercury emissions from power plants, which are among the principal pollutants under the EPA regulation at issue in this case. EPA and other amici will demonstrate more fully the legal basis for EPA's decision, but to put the specific arguments regarding mercury exposure in perspective, this brief will first outline the legal framework that EPA properly followed here.[2]

The provisions of the 1990 amendments to the Clean Air Act that addressed hazardous air pollutants, 42 U.S.C. § 7412, significantly changed the regulation of those pollutants. In subsection 7412(b), Congress listed more than 180 substances which EPA was required to treat as hazardous air pollutants and to regulate accordingly. Subsection 7412(c) requires EPA to issue regulations, pursuant to the standard-setting criteria of subsection 7412(d), for all industrial sources of hazardous air pollutants on the list. Paragraph (3) of subsection 7412(d) requires that the reduction in emissions for existing sources "shall not be less stringent, and may be more stringent than - **(A)** the average emission limitation achieved by the best performing 12 percent of the existing sources [subject to certain qualifications not relevant here] with the lowest achievable emission rate applicable to the [relevant] source category "

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[2] For a useful pre-2011 history of EPA's efforts to regulate mercury and other power plant emissions, see Keith Harley, *Mercurial but Not Swift: U.S. EPA's Initiative to Regulate Coal Plant Mercury Emissions Changes Course Again as its Enters a Third Decade*, 86 Chi.-Kent. L. Rev. 277 (2011).

There is no requirement that EPA consider cost in determining this minimum stringency "floor" for power plants because, almost by definition, if "the best performing 12 percent" of such sources is already complying, the cost must be reasonable. However, if EPA wishes to impose more stringent emission reduction requirements than those being achieved by the best performing sources in a category, then subsection 7412(d)(2) does require it to consider costs.

Subsection 7412(c) does not contain an express exception for power plants, whose emissions of other pollutants, including sulfur dioxide and oxides of nitrogen, are subject to regulation under the acid rain control provisions of the 1990 Clean Air Act amendments, 42 U.S.C. §§ 7651-7651*o*. However, subsection 7412(n) has special provisions relating to power plants, referred to there as "electric utility steam generating units." Subparagraph (1)(A) required EPA to produce a study within three years from the date of enactment of the 1990 amendments "of the hazards to public health reasonably anticipated to occur" as a result of their emissions of the hazardous air pollutants listed under subsection (b) which includes mercury compounds. The study was to be sent to Congress, and it was to include "alternative control strategies for emissions which may warrant regulation under this section." Thereafter, the "Administrator shall regulate electric utility steam generating units under this section, if the Administrator finds such regulation is appropriate and necessary after considering the results of the study required by this

subparagraph." Of significance to this case, there is no mention of costs anywhere in section 7412 in the directions for the study or in the mandate to regulate based on the study's findings.

Other provisions of subsection 7412(n) reenforce EPA's position that the decision to regulate emissions from power plants of hazardous air pollutants listed under subsection 7412(b) was to be made without regard to costs. Subparagraph (1)(B) mandates a separate study to be done by EPA regarding mercury emissions from power plants (but without a mandate to regulate), for which it was given four years. Three aspects of that study are significant: the study was to include not only power plants, but also "municipal waste combustion units, and other sources, including area sources," the latter term being defined in subsection 7412(a)(2). Second, Congress required that this study cover not only health effects, but also environmental effects. Third, and in contrast to subparagraph (1)(A), subsection (1)(B) required EPA to consider the "technologies which are available to control such emissions, and the *costs* of such technologies." (Emphasis added.) The significance of the addition of environmental effects and the necessity for considering cost in the broader study under subparagraph (B), but not the study on which the regulation at issue is based under subparagraph (A), is underscored by subparagraph (C). That provision directs the National Institute of Environmental Health Sciences to conduct a study to determine the threshold level of mercury exposure below which human adverse health effects are not expected to

occur. That study, which was also required to include a threshold for mercury concentrations from the tissue of fish consumed by humans, covers human health effects only and, like the study under subparagraph (A), makes no mention of costs.

Similarly, subparagraph 7412(n)(2)(A) mandates a study of coke oven emissions, this one within six years. It was to be done by EPA and the Department of Energy "to assess coke oven production emission control technologies and to assist in the development and commercialization of technically practicable and economically viable control technologies which have the potential to significantly reduce emissions of hazardous air pollutants from coke oven production facilities." There is no mandate to regulate if certain findings are made, but rather under subparagraph (C), the study is to make "recommendations to the Administrator identifying practicable and economically viable control technologies for coke oven production facilities to reduce residual risks remaining after implementation of the standard under subsection (d) of this section." These recommendations must include "economically viable" solutions for coke ovens, in contrast to the exclusion of all specific references to costs under subsection 7412(n)(1)(A) for hazardous air pollutant emissions from power plants. Therefore, the specific inclusions of costs in other parts of subsection 7412(n) remove all doubt that Congress intended that EPA's decision whether to regulate emissions of hazardous air pollutants, including mercury emissions, from power plants on the basis

of the results of the study mandated by subsection 7412(n)(1)(A), should be made without regard to the costs that such basic regulation might impose. That result is not only clear from the statute itself, but makes eminent sense given the dangers to human health from exposure to hazardous air pollutants such as mercury, and through methylmercury a well-documented neurotoxin that poisons brain development in the fetus, and to which humans are exposed in the food supply through fish.

ARGUMENT

MERCURY EXPOSURE FROM POWER PLANT EMISSIONS PRESENTS HIGHLY SIGNIFICANT AND SERIOUS RISKS TO HUMAN HEALTH.

This brief will focus specifically on the public health implications of reducing (or failing to reduce) toxic air pollutants– most significantly mercury – under the Mercury and Air Toxics Standards (MATS) for power plants. Mercury is one among a number of toxic air contaminants that will be reduced in consequence of this rule; others include arsenic, beryllium, cadmium, chromium, cobalt, lead, manganese, nickel and selenium. Several other pollutants emitted by power plants (most notably $PM_{2.5}$, SO_2, NO_x, HCl and CO_2) also have serious public health impacts. Although this brief seeks to inform the Court about the significant public health impacts of mercury – and consequent benefits from the MATS rule due to

mercury reduction alone, the rule being challenged also produces other public health benefits by reducing emissions of toxics that are known human carcinogens (arsenic, beryllium, cadmium. chromium and nickel), or of substances reasonably anticipated to be human carcinogens (cobalt, lead and selenium), neurotoxicants (arsenic, lead, and manganese), and possible diabetogenes.[3]

METHYLMERCURY FROM COAL FIRED POWER PLANTS

The principal documents regarding the health effects of exposure to mercury are the EPA's Mercury Study Report to Congress (1997) ("EPA Mercury Study"),[4] EPA's Utility Hazardous Air Pollutant Report to Congress (1998) ("Hazardous Pollutant Report"),[5] and the National Research

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[3] Knowledge of the harmful effects of other pollutants besides mercury has increased since 1990. For example, arsenic recently has been linked to type 2 diabetes. HHS, 13th Report on Carcinogens, National Toxicology Program (2013); Grandjean P. *et al.*, Developmental Neurotoxicity of Industrial Chemicals, *Lancet* 368(9553):2167-2178 (2006); Navas-Acien A., *et al.* Arsenic Exposure and Type 2 Diabetes: A Systematic Review of the Experimental and Epidemiologic Evidence. *Environ. Health Perspect.* 114:641–648 (2006); Maull E. A., *et al.* Evaluation of the Association between Arsenic and Diabetes: A National Toxicology Program Workshop Review. *Environ. Health Perspect,* http://dx.doi.org/10.1289/ehp.1104579, Online 10 August 2012.

[4] Available at http://www.epa.gov/mercury/report.html.

[5] Available at http://www.epa.gov/airtoxics/combust/utiltox/utoxpg.html#TEC.

Council's Toxicological Effects of Methylmercury (2001) ("NRC Effects of Methylmercury").[6] Together they demonstrate that mercury is a ubiquitous environmental toxicant and that coal-burning electric utilities are the largest source category of anthropogenic mercury emissions, accounting for one half of emissions in the United States. *See* 77 Fed. Reg. 9310 (2012) (column 2). These reports also show that the evidence that environmental emissions of mercury have significant negative impacts on population health in the U.S. has advanced considerably since the time of the 1990 Clean Air Act amendments which mandated EPA's study.

As explained in these three studies/reports, mercury is a toxic metal released into the environment from both natural and industrial sources. Coal-fired power plants are the largest remaining anthropogenic source of mercury emissions in the U.S. As coal burns, its mercury content is emitted into the air, transported through the atmosphere and then readily deposited (particularly with the rain) onto land and into water bodies including streams, lakes, and oceans. Human exposure to mercury originating from power plant emissions is mostly via a form of organic mercury called methylmercury. In aquatic ecosystems, bacteria in sediments transform inorganic mercury from power plant emissions into methylmercury, which then bioaccumulates in the aquatic food chain resulting in high mercury

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[6] National Research Council. Toxicological Effects of Methylmercury (National Academies Press, 2001).

concentrations in the tissue of higher trophic level (*i.e.*, predatory) fish consumed by humans.[7] Fish accumulate mercury from their food as they grow, as they cannot eliminate it. Older and larger fish have higher mercury content. Thus, human exposure to mercury from power plants is indirect; humans are exposed to methylmercury through consumption of contaminated fish.

Each year, 2,820 pounds of mercury are discharged into the Nation's waters.[8] EPA has concluded that mercury pollution from coal-fired steam electric plants may be making fish unsafe for human consumption in 65% of the waters that receive discharges. *Id.*, Mercury is far and away the leading cause for EPA and States to issue more than 4000 fish-consumption advisories, warning fishers "do not eat" or "limit consumption" of some or all fish from over 40% of lake acres and river miles and most of the continental U.S. coastline.[9] Moreover the number of mercury-based advisories

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[7] Agency for Toxic Substances and Disease Registry, Toxicological Profile for Mercury-Potential for Human Exposure (1999) at http://www.atsdr.cdc.gov/toxprofiles/tp.asp?id=115&tid=24.

[8] EPA, Technical Fact Sheet: Trends in Blood Mercury Concentrations among Women of Reproductive Age (2013) at http://water.epa.gov/scitech/swguidance/fishshellfish/fishadvisories/upload/Technical-Fact-Sheet-Trends-in-Blood-Mercury-Concentrations-among-Women-of-Childbearing-Age.pdf.

[9] EPA, National Listing of Fish Advisories: Technical Fact Sheet 2010 (2012). http://water.epa.gov/scitech/swguidance/fishshellfish/fishadvisories/technical_factsheet_2010.cfm.

grew steadily from early-1990s through 2010 (latest year of data). Studies of past efforts to reduce mercury emissions from coal-fired boilers have shown that when these emissions are reduced, concentrations in fish immediately downwind from plants in both Massachusetts and Florida have reduced; the Florida study used isotopic labels to trace the mercury to coal burning plants.[10]

While mercury emissions from U.S. power plants have the highest deposition in areas close to the plants, they also are of global concern due to the atmospheric transport. Using isotopic labeling, mercury in the Arctic has been clearly traced to coal burning emissions not only from the U.S. and the rest of North America but also from Asia, Russia and Europe.[11] Mercury is of such global concern that nations have negotiated, with the U.S. as the first cosignatory, the United Nations Minamata Convention on Mercury. The Minamata Convention "recognizes that mercury is a chemical of global concern owing to its long-range atmospheric transport, its persistence in the

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[10]Hutcheson M.S., et al. Temporal and Spatial Trends in Freshwater Fish Tissue: Mercury Concentrations Associated with Mercury Emissions Reductions, *ES&T* 48:2193-2202 (2014); Atkeson, T. D., *et al.* Recent Trends in hg Emissions, Deposition, and Biota in the Florida Everglades: A Monitoring and Modeling Analysis. In: Dynamics of Mercury Pollution on Regional and Global Scales: Atmospheric Processes, Human Exposure around the World, Springer Publisher, Norwell, MA, 26: 637-656. (2005).

[11] Dumford D., *et al.* Long Range Transport of Mercury to the Arctic and across Canada, Atmos. Chem. Phys. 10:13; 6-63-6083 (2010).

environment once anthropogenically introduced, its ability to bioaccumulate in ecosystems and its significant negative effects on human health and the environment" Article 8 of this Convention includes an agreement among all nations to "...control[] and, where feasible, reduc[e] emissions of mercury and mercury compounds, often expressed as 'total mercury', to the atmosphere..." Coal-fired power plants are listed as among the sources that are to be controlled under this regime. The Convention was negotiated well after the adoption of the Clean Air Act, but it demonstrates that not only Congress and the EPA but also governments and environmental authorities globally recognize the serious public health impacts of human-caused (anthropogenic) mercury releases generally, and emissions from coal-fired power plants specifically. Developing countries are contributing increasingly to global emissions from coal-fired power plants. If the regulation at issue in this case were set aside, it could provide cover to other countries for failing to take action to implement Article 8. Thus, not only will the benefits to public health that will result from reductions in mercury exposures under the MATS be substantial, but the implications of overturning this rule may encourage other countries to back away from this agreement, with global consequences which in themselves could increase methylmercury exposure to the U.S. population.

Both freshwater and ocean species of fish can contain high levels of mercury. Apex predator fish species, marine species such as shark, tuna, and swordfish, and freshwater species such as

bass, pickerel and wall-eye, which are at the top of the food chain, and some marine mammals are of particular concern for causing high human exposure levels because they accumulate the highest levels of mercury. Individuals or populations who consume large amounts of these types of foods are more highly exposed and have been found to be disproportionately affected by adverse health effects, mainly neurologic outcomes caused by mercury toxicity.

METHYLMERCURY TOXICITY

Mercury has long been recognized as neurotoxic (toxic to the nervous system), but only in recent decades has methylmercury, at levels found in the ambient environment, been recognized as a neurodevelopmental toxicant, *i.e.*, toxic to the developing central nervous system. [12] It is also suspected to be cardiotoxic, *i.e.*, toxic to the cardiovascular system.[13] At this time, there is no

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[12] National Research Council, *supra*, note 6, pp. 174-202; Grandjean, P. *et al.* Cognitive Deficit in 7-year-old Children with Prenatal Exposure to Methylmercury, *Neurotoxicol Teratol.* 19, 417–428 (1997).

[13] Roman, H. A. *et al.* Evaluation of the Cardiovascular Effects of Methylmercury Exposures: Current Evidence Supports Development of a Dose-Response Function for Regulatory Benefits Analysis, *Environ. Health Perspect* 119, 607–614 (2011); Karagas, M. R. *et al.* Evidence on the Human Health Effects of Low-Level Methylmercury Exposure. *Environ. Health Perspect.* 120, 799–806 (2012); Rice, G. E., *et al.* A Probabilistic Characterization of the Health Benefits of Reducing Methylmercury Intake in the United States. *ES&T* 44, 5216–5224 (2010); Guallar, E. *et al.* Mercury, Fish Oils,

evidence for a threshold below which neurodevelopmental effects do not occur.[14] Such a threshold is often referred to as a "reference dose" or RfD, a health standard that incorporates not only the evidence for a threshold but also the level of uncertainty around the threshold. This means that it is reasonable to believe that any reductions in exposure that can be achieved will have benefits across the population. Even at low exposure levels, methylmercury can lead to reductions in IQ for developing children.[15] These deficits in IQ may not be clinically apparent in individual children, but on a population level they have cumulative impacts with large public health and economic consequences.

Precise quantification of the risks of methylmercury exposure and the benefits of exposure reduction have been complicated by the confounding in the relationship between dietary methylmercury exposure and DHA, a beneficial omega three fatty acid found in fish oil.[16] This confounding means that some of the negative health effects of methylmercury are apparently offset by the benefits attributable to increased intake of DHA; it also means that methylmercury is attenuating the benefits of DHA consumption to

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and the Risk of Myocardial Infarction. *N. Engl. J. Med.* 347, 1747–1754 (2002).

[14] Karagas, *supra*, note 13; Rice *supra*, note 13.

[15] Karagas, *supra*, note 13; Goldman, L. R., *et al.*, Technical Report: Mercury in the Environment: Implications for Pediatricians. *Pediatrics* 108, 197–205 (2001).

[16] Rice, *supra*, note 13.

pregnant women who eat contaminated fish. Thus, fish advisories that control mercury exposure via limiting the consumption of fish for women of childbearing age and for children invariably decrease these populations' intake of beneficial DHA and other omega three fatty acids, possibly harming other adults in the household when fish is less available in household meals.[17] Where possible, it is preferable to reduce mercury exposure, such as by limiting emissions from power plants. The most recent scientific studies, which have simultaneously accounted for methylmercury's hazardous effects and the benefits of DHA, have demonstrated that methylmercury is even more toxic than originally suspected. [18]

Methylmercury is also neurotoxic to adults. There are now a number of reports of methylmercury poisoning in adults occurring with regular consumption of methylmercury-contaminated fish. Adults with methylmercury poisoning have had severe and debilitating symptoms: paresthesia, ataxia, weakness, vision and hearing impairment, muscle tremor and

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[17] Rheinberger, C. M., *et al*, Risk Tradeoffs in Fish Consumption: A Public Health Perspective, *ES&T* 46: 12337-12346 (2012); Rheinberger, C. M., *et al.*, The Welfare Value of FDA's Mercury in Fish Advisory: A Dynamic Reanalysis, *J. Health Econ.* 37: 113-122, (2014).

[18] Oken E, *et al.* Maternal Fish Intake during Pregnancy, Blood Mercury levels, and Child Cognition at Age 3 Years in a US Cohort, *Am. J. Epidemiol.* 167, 1171 (2008); Lederman SA, *et al.* Relation Between Cord Blood Mercury Levels and Early Child Development in a World Trade Center Cohort, *Environ. Health Perspect.* 116:1085 (2008).

spasticity and even coma or death.[19] A recent article reviewed 25 clinical cases of methylmercury poisoning among adult fish eaters. [20] It reported variable exposure levels (7 to 125 µg/L of mercury in blood) in association with toxicity from mercury in fish. Further, there is no medical treatment for methylmercury exposure other than to eliminate consumption of mercury contaminated fish and to wait until blood levels fall to a lower level. Chronic lower levels of exposure to methylmercury have been found to cause neurological impairment in adults. Asymptomatic adults with methylmercury levels above the WHO standard established for children (see below) scored significantly lower on tests of fine motor speed and neurocognitive tests.[21]

Cardiovascular effects of methylmercury exposure have emerged as a concern even at exposure levels below current levels of concern.[22] Evidence also suggests that mercury exposure is a risk factor for myocardial infarction and possibly other cardiovascular effects.[23] Rice et al. in 2010 additionally pointed out the importance of including cardiovascular effects in estimating the value of health benefits achieved by reducing

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[19] Goldman, *supra*, note 15.

[20] Silbernagel, S. M. *et al.* Recognizing and Preventing Overexposure to Methylmercury from Fish and Seafood Consumption: Information for Physicians. *J. Toxicol.* 2011, 1–7 (2011).

[21] Yokoo, E. M. *et al.*, Low Level Methylmercury Exposure Affects Neuropsychological Function in Adults. *Environ. Health Glob. Access Sci. Source* 2, 8 (2003).

[22] Roman, *supra*, note 13; Karagas, *supra*, note 13.

[23] Roman, *supra*, note 13.

methylmercury exposure, and they concluded that not including cardiovascular effects in benefit estimations leads to significant underestimation of the public health benefits that can be achieved by reducing mercury emissions.[24]

As of 2001, the EPA had identified 0.1 µg /kg-day as a reference dose (RfD) for methylmercury; this is an exposure level of 5.8 µg/L methylmercury in umbilical cord blood, the guideline recommended by the National Research Council. [25] Methylmercury crosses the placental barrier and concentrations found in newborn cord blood are actually about 1.7 times higher than maternal blood levels. [26] Therefore, the EPA RfD is equivalent to 3.5 µg/L methylmercury in maternal blood. In 2004, based on the same studies, but different estimates of uncertainty, the WHO established a weekly limit, or provisional tolerable weekly intake (PTWI) of 1.6 µg/kg-week.[27]

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[24] Rice, *supra*, note 13.

[25] EPA. Integrated Risk Information System-Methylmercury (MeHg) (CASRN 22967-92-6) (2001); http://www.epa.gov/iris/subst/0073.htm.; Mahaffey, K. R., *et al.* Adult Women's Blood Mercury Concentrations Vary Regionally in the United States: Association with Patterns of Fish Consumption (NHANES 1999–2004), *Environ. Health Perspect.* 117, 47–53 (2009).

[26] Stern AH, *et al.* An Assessment of the Cord Blood: Maternal Blood Methylmercury Ratio: Implications for Risk Assessment, *Environ. Health Perspect.* 111:1465-70 (2003); Yokoo, *supra*, note 21.

[27] Joint FAO/WHO Expert Committee on Food Additives. In: Sixty-first Meeting, Rome, 10–19 June 2003: Summary and Conclusions, Food and Agriculture Organization of the

Since these guidelines were developed, new evidence has emerged that indicates that there are people in the population with increased genetic susceptibility to methylmercury toxicity.[28] Moreover, as noted above, the most current evidence on the health effects of mercury suggests that no threshold can be identified.[29] That evidence is strongest for the neurodevelopmental effects. Karagas et al.'s review published in 2012 shows that both neurodevelopmental effects and cardiovascular effects occur at exposure levels below the levels recorded in the studies used to set the EPA reference dose.[30] Rice et al. assumed a 90% probability of a linear no threshold dose-response for neurodevelopmental toxicity in their benefits model because they "find no strong biological support for this population threshold".[31] Additionally, the Karagas study shows that harms from mercury exposure can occur even with infrequent consumption of seafood.[32] In other words, the assumption of a threshold or a "safe" level, as implied by an RfD or a PTWI, probably underestimates the impacts of mercury on population health.

United Nations & World Health Organization; 2003. Available at: ftp://ftp.fao.org/es/esn/jecfa/jecfa61sc.pdf.

[28] Julvez J., et al., Genetic Susceptibility to Methylmercury Developmental Neurotoxicity Matters, *Frontiers in Genetics*, 4; 278;1-4 (2013).

[29] Karagas, *supra*, note 13; Rice, *supra*, note 13.

[30] Karagas, *supra*, note 13.

[31] Rice, *supra*, note 13 at pp. 5218.

[32] Karagas, *supra*, note 13.

METHYLMERCURY EXPOSURES IN THE U.S.

Numerous studies have demonstrated that the developing brain is most sensitive to methylmercury's neurotoxic effects and thus the focus has been on exposure to women of child bearing age and young children. In 2004 the CDC reported that 5.7% of women of childbearing age had blood mercury levels at or above the EPA reference dose for newborns in the 1999-2002 NHANES survey.[33] Blood mercury levels have been tracked over time and the level reduced sharply to 3.14% in 2001-2002, and leveled off to around 2.1% thereafter. These same data show no downward trend in consumption of fish and seafood.[34] Thus even with reductions in mercury from past regulation, as well as advice to consume fish and seafood with lower mercury levels, a significant percentage of the population has mercury exposures that are too high for children and women of childbearing age.

Because of variable emissions levels in different regions of the U.S., variable levels of methylmercury in different water bodies, variable uptake and bioaccumulation of methylmercury in different species of fish, and variable fish

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[33] Centers for Disease Control, Blood Mercury Levels in Young Children and Childbearing-aged Women --- United States, 1999—2002, *MMWR*. 53(4); 1018-1020, November 5, 2004.

[34] EPA, Trends in Blood Mercury Concentrations and Fish Consumption Among U.S. Women of Childbearing Age NHANES, 1999-2010, Final Report EPA-823-R-13-002. (2013).

consumption by different communities and ethnic groups, some populations are at greater risk of methylmercury toxicity than others.[35]

Mercury concentrations are highest in large apex predator fish and other large fish. While all fish consuming members of the population are at risk, a disproportionate burden of exposure and neurodevelopmental and cardiovascular health effects fall on specific subpopulations who consume more of such fish or who habitually eat fish from contaminated areas. Sensitive populations in the U.S. include coastal populations, Asian and Pacific Islander populations, Native American groups and populations who consume more fish as an effort to have a more healthful diet.[36] Health-conscious people may replace all red meat dishes with fish, consuming fish at one or more meals per day, ironically jeopardizing their health. Fish populations downwind from power plant emissions globally, accumulate methylmercury because of long-range transport of mercury in air. However, higher rates of mercury deposition have been found in areas very close to power plant emissions. EPA has focused its benefits assessment on consumption of fish from such locations among recreational anglers who frequent these areas

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[35] Mahaffey, *supra*, note 25; Sheehan, M. C. *et al.* Global Methylmercury Exposure from Seafood Consumption and Risk of Developmental Neurotoxicity: a Systematic Review, *Bull. World Health Organ.* 92, 254–269F (2014); Schober, S, *et al.*, Blood Mercury Levels in US Children and Women of Childbearing Age, 1999-2000, *J. Am. Med. Assoc.* 289 (13):1667-1674 (2003).

[36] Sheehan, *supra*, note 35.

because of the availability of data on fishing licenses and localized fish contamination levels. There has been less information about the extent to which contaminated fish from such areas immediately downwind of power plants are entering commerce, nor is much known about exposures experienced by subsistence fishers in those regions.

Underscoring the importance of understanding subpopulation vulnerabilities, a 2003 study found that some children in middle to upper income families in San Francisco had methylmercury levels 40 times the national average for that age group because of fish and seafood consumption.[37] That study assessed hair mercury from NHANES 1999-2000 data and found that mercury levels were on average three fold higher for women and two fold higher for children who were identified as frequent fish consumers in a food frequency questionnaire.[38] A 2009 study using 1999-2004 NHANES data found that women who lived in the Northeast and in coastal regions of the U.S. were more likely than residents of other regions to have mercury levels exceeding 3.5 μg/g, the concentration (as noted above) considered to be an appropriate level of concern because it takes

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[37] Hightower and Moore, "Mercury Levels in High-End Consumers of Fish," *Environmental Health Perspectives* 111, (4): 604–8. (2002) doi:10.1289/ehp.5837.

[38] McDowell et al., "Hair Mercury Levels in U.S. Children and Women of Childbearing Age: Reference Range Data from NHANES 1999-2000," *Environmental Health Perspectives* 112; (11): 1165–71, (2004) doi:10.1289/ehp.7046.

into account the concentration of methylmercury across the placenta and more accurately reflects the amplification of exposure to the fetus.[39]

A systematic review conducted for the WHO/FAO also showed that human methylmercury levels vary globally depending on geographical location and fish consumption habits.[40] It found that coastal populations and Artic populations (including those in Alaska) have mercury concentrations that were elevated above other subpopulations and that many have exposures above the WHO reference level. Coastal regions of the world have the largest number of at risk individuals for neurodevelopmental and cardiovascular effects due to methylmercury exposure. The WHO report concluded that a large number of individuals worldwide, including in the United States, are exposed to mercury at levels above the WHO recommended level.

According to the EPA's 2014 report, those with the highest per capita fish consumption were either of lower income (subsistence consumers) or higher income groups and resided either in the coastal Northeast or on the West Coast.[41] Differences in average intakes are substantial; consumption rates nearly double between the 50th and 75th percentile for most groups. Asian

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[39] Mahaffrey, *supra*, note 25.

[40] Sheehan, *supra*, note 35.

[41] EPA, Estimated Fish Consumption Rates for the U.S. Population and Selected Subpopulations (NHANES 2003-2010); Final Report EPA-820-R14-002.. pp. 49–92 (2014).

populations also have higher exposures to methylmercury. According to the CDC, in 2011-2012 when the general population geometric mean (GM) methylmercury in blood was 0.5 μg/L, the mean for Asian Americans was 1.58 μg/L.[42] Overall 15.8% of Asian Americans were at or above the EPA level of concern (based on *in utero* exposure) of 5.8 μg /L, compared to 2.8% of non-Hispanic Whites, 2.15 of Non-Hispanic Blacks and 1.3% of Hispanics.[43]

The United States is the world's third largest emitter of mercury behind China and India, and fossil fuel combustion is the largest source.[44] Moreover, given that much of the harm from mercury releases occurs locally, the emissions from sources in the US are more harmful to the US population than those from China and India. While there are natural sources of mercury in water, and therefore in fish, these human-caused (anthropogenic) emissions are causing health risks that are additive to natural levels and inputs from other sources and are therefore increasing health risks significantly.

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[42] CDC, Fourth National Report on Human Exposure to Environmental Chemicals Updated Tables (2014), pp. 189-194
http://www.cdc.gov/exposurereport/pdf/fourthreport_updatedtables_aug2014.pdf.

[43] Mortensen M.E., *et al.* Total and Methyl Mercury in Whole Blood Measured for the First Time in the U.S. Population: NHANES 2011–2012, *Environ. Res.* 134:257-264 (2014).

[44] EPA, ORA Mercury Emissions: The Global Context. (2014) available at http://www2.epa.gov/international-cooperation/mercury-emissions-global-context.

CONCLUSION

When Congress enacted the Clean Air Act Amendments of 1990, there was ample evidence that mercury was harmful to human health, but Congress nevertheless mandated further study before permitting EPA to regulate mercury emissions from power plants. That study has been completed and, along with much other research, has removed all doubts about the harmful health (and environmental) effects of mercury and the large public health benefit of reducing mercury along with arsenic and other pollutants. Under those Amendments, that set of findings is all EPA needed to impose the regulations that it did, and accordingly the decision of the Court of Appeals should be affirmed.

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March 4, 2015

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